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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER
LEFFERS JR, GERALD G

ART UNIT	PAPER NUMBER
1636	21

DATE MAILED: 08/13/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/800,520	IBA ET AL.	
	Examiner	Art Unit	
	Gerald G Leffers Jr., PhD	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *07 July 2003* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *34-45* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *34-45* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☒ Certified copies of the priority documents have been received in Application No. *09/214,465* .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) *15* .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other:

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 5/29/03 as Paper No. 17 has been entered.

Several claims were amended in Paper No. 17 (claims 34-36, 38-39) and several new claims were added (claims 40-45). Claims 34-45 are pending and under consideration in the instant application.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 34-45 are is rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Claims 34-45 are directed to a broad class of vectors and methods of use thereof to express desired genes in a given cell. At least two of the claims are directed to use of the vectors

in gag/pol expressing cells. In the specification, however, the short-lived transcript drug resistance genes are only described in the context of selecting cells comprising very specific expression constructs (e.g. construct A, pages 8-12) integrated into the host cell genome for the purpose of preparing pre-packaging cells useful for the preparation of retroviral gene transfer vectors (e.g. pages 16-17, bridging paragraph; page 22, 1st paragraph). No broader use for the recited short-lived transcript drug resistance genes is contemplated in the originally filed specification or claims. Thus, the broader scope of use encompassed by the rejected claims is impermissible NEW MATTER.

Response to Arguments

Applicant's arguments filed in Paper No. 17 have been fully considered but they are not persuasive. The response essentially argues the specification is not limited to just packaging cell types because construct (A) (see pages 8-12) does not require an LTR or packaging signal of a retrovirus genome (i.e. in contrast to construct (B)). The response asserts that construct (A) can be used in any cell type to express any gene product. The response also asserts that claims 40 and 45, which specify that the host cell comprising the construct is a gag/pol expressing cell overcomes the grounds of rejection.

The rejection is made on the grounds that the invention is described only in the context of specific vectors (i.e. construct A) in a particular cell type (i.e. gag/pol producing cells). There is no literal support in the originally filed specification for the broader range of constructs and methods embraced by the amended claims, and the rejection was made in terms of NEW MATTER. The examiner does not question the enablement of the claimed invention by the specification, just whether there is support for that which is now claimed. There is not. Claims

40 and 45 would be allowable if amended to include the minimum recited elements of construct A as defined in the specification.

Claims 35-40 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter that was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The rejected claims comprise the limitation "...wherein said expression vector confers drug resistance when transfected into a cell and the drug resistance gene is transcribed at a higher rate under selection with the drug because of the presence of the mRNA-destabilizing sequence." This limitation specifies that the presence of the short-lived transcript drug resistance marker *necessarily* confers on the expression vector an increased expression of a desired gene. There is no literal support for this concept in the specification. The specification provides support for *selection* from a larger population of cells comprising the construct A, a subpopulation wherein the expression of the short-lived transcript is elevated above the remaining population. Therefore, the cited limitation is impermissible NEW MATTER.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

The rejected claims are drawn towards expression vectors comprising a drug resistance gene as a selection marker and an mRNA destabilizing sequence (e.g. claim 34). The vector can be one intended to "highly express" gene products encoded by the vector through transfection of host cells with the vector and selection of cells comprising the vector with the drug (e.g. claim 35). The mRNA destabilizing sequence can be obtained from the untranslated region of a gene encoding c-fos (e.g. claim 36). Cells comprising the expression vector are claimed (claim 38) as well as a method of producing cells for expressing gene products encoded by the expression vector comprising transforming the host cells with the vector (claim 39).

Claims 34-38, 41-43 are rejected under 35 U.S.C. 103(a) as being unpatentable over Pavlakis et al (U.S. Patent No. 5,972,596; see the entire patent) in view of DePonti-Zilli et al (PNAS USA 1988, Vol. 85, pages 1389-1393; see the entire reference). **This rejection is maintained for reasons of record in Paper No. 14, mailed 2/12/03 and repeated below.**

The Pavlakis patent (the '596 patent) teaches methods for identifying and correcting inhibitory/instability sequences (INS) within the coding region of an mRNA of a desired protein

such that the level of production of the desired protein can be increased (e.g. Abstract; columns 5-6, bridging paragraph). Pavlakis et al teach that in order to evaluate whether putative regulatory sequences are sufficient to confer mRNA stability control (e.g. destabilization) on an mRNA transcript, DNA sequences coding for the suspected INS region are fused to an indicator (or reporter) gene to create a gene coding for a hybrid mRNA. The DNA sequence fused to the indicator gene can be cDNA, genomic DNA or synthesized DNA. Examples of acceptable reporter genes known in the art are genes encoding neomycin resistance protein (note: neomycin itself is not a protein), B-galactosidase, chloramphenicol resistance, luciferase, B-globin, PGK1 and ACT1.

The '596 patent teaches that the stability and/or utilization of the mRNAs generated by fusion of the indicator genes and sequences suspected of encoding an INS region is tested by transfecting the hybrid genes into host cells which are appropriate for the expression vector used to clone and express the mRNAs. The resulting levels of mRNA are determined by standard methods of determining mRNA stability (e.g. Northern blots, S1 nuclease mapping or PCR methods), and the resulting levels of protein produced are quantitated by protein measuring assays (e.g. ELISA, western blot, etc.). The INS regions are identified by a decrease in the protein expression and/or stability of the hybrid mRNA as compared to the control indicator RNA (e.g. column 13, lines 44-62). Once INS regions of a particular target gene are identified, the coding sequence can be altered such that the expressed polypeptide is the same one encoded by the original coding sequence, or a conservative variant of the original polypeptide (e.g. column 16, section 3). Mutated or altered coding sequences designed to remove INS sequences

are then tested in the same manner as was used to identify the INS sequence (e.g. column 16, lines 45-56).

The Pavlakis et al patent teaches that genes encoding or suspected of encoding mRNAs containing inhibitory/instability regions within the coding region are particularly relevant to the invention (column 12, lines 34-36). In particular, c-fos is identified as a protein whose coding sequence is known in the art to comprise INS sequences that result in the c-fos transcript being unstable such that it is rapidly degraded (e.g. column 2, lines 8-13; column 12, lines 15-35). Example 3 is directed towards an embodiment wherein fragments encoding c-fos are operatively linked to a sequence encoding a reporter protein (i.e. RSV gag).

The Pavlakis et al patent does not exemplify an embodiment where the neomycin resistance gene is operatively linked to a coding sequence comprising an INS, although it does suggest that the neomycin resistance gene would be an effective reporter in their system. The '596 patent doesn't explicitly teach the fusion of a coding sequence for neomycin resistance to any part of the c-fos gene.

The DePonti-Zilli et al reference teaches the characterization of a 40 base-pair sequence in the 3' end of the B-actin gene with regard to regulating B-actin mRNA transcription during myogenesis (e.g. Abstract). DePonti-Zilli et al teach that fusion of the 40 base-pair sequence 3' to the genes for a-cardiac-actin and neomycin-resistance protein confers the B-actin mRNA regulatory pattern on the hybrid constructs when introduced into C2C12 cells (e.g. Abstract; Figure 3). Hybrid transcript levels were detected by S1 nuclease protection assays using end-labeled neomycin resistance gene probes (e.g. page 1389, column 2, "RNA Isolation and Nuclease S1 Analysis"; Figure 3). The authors conclude that although the 40 base-pair sequence

from B-actin fused to the neomycin resistance coding sequence conferred B-actin transcriptional regulatory patterns on the hybrid transcript, the control was not at the level of RNA stability (e.g. pages 1392-1393, bridging paragraph). Therefore, DePonti-Zilli et al do not teach the construction and use of a short-lived transcript drug resistance gene.

It would have been obvious to one of ordinary skill in the art at the time of the invention to use the neomycin resistance gene in the methods of Pavlakis et al as an indicator to identify instability sequences (INS) of a gene encoding a transcript known or suspected to possess such INS sequences because Pavlakis et al teach it is within the skill of the art to use the gene encoding neomycin resistance as a reporter gene to identify such instability sequences and because DePonti-Zilli et al teach the use of the neomycin resistance gene to characterize a putative transcriptional regulatory sequence when the putative regulatory sequence is fused to the sequence encoding neomycin resistance. One would have been motivated to do so in order to receive the expected benefit, as suggested by Pavlakis et al and actually exemplified by DePonti-Zilli et al, of being able to characterize the ability of a putative transcriptional regulatory sequence to affect the stability/utilization of a neomycin resistance gene transcript. Absent any evidence to the contrary, there would have been a reasonable expectation of success in utilizing the neomycin resistance gene as a marker to identify transcriptional regulatory sequences that destabilize the neomycin resistance transcript. In cases in which such a destabilizing element is identified using the neomycin resistance gene as a reporter, one would necessarily have generated a short-lived transcript drug resistance gene.

The limitation of a cell transformed with the vector of claim 35 and selected with the corresponding drug does not necessarily confer any structural/functional distinction on the cell

that would not be present in the cells taught by Schuler. Differences in gene expression for the constructs taught in the specification appear to be due to the location of insertion of the targeting vector in the specification and not due to some structural aspect of the claimed vector itself.

Claims 34-38, 41-43 are rejected under 35 U.S.C. 103(a) as being unpatentable over Pavlakis et al (U.S. Patent No. 5,972,596; see the entire patent) in view of Gritz et al (Gene 1983, Vol. 25, pages179-188). **This rejection is maintained for reasons of record in Paper No. 14, mailed 2/12/03 and repeated below.**

The teachings of the Pavlakis et al patent (the '596 patent) are described above and applied as before, except: the Pavlakis et al patent does not explicitly teach an embodiment where the hygromycin resistance gene is operatively linked to a coding sequence comprising an INS.

Gritz et al teach the cloning and characterization of the plasmid-borne gene (hph) encoding hygromycin B phosphotransferase from E. coli (e.g. Abstract). Gritz et al teach that when placed in the appropriate shuttle vector, hph allows for direct selection of cells comprising the shuttle vector in yeast as well as for E. coli (e.g. Abstract). This selection varies for different concentrations of hygromycin B and different constructs comprising different transcription initiation points (e.g. Figure 5 & Table II). Thus, Gritz et al teach that different levels of hygromycin B phosphotransferase in a cell expressing the hph gene can be detected by genetic selection. In addition to detecting the levels of hygromycin B phosphotransferase by direct genetic selection, Gritz et al teach a method for directly assaying enzyme activity in a cell extract (e.g. page 181, column 1, last paragraph; Table 1).

It would have been obvious to one of ordinary skill in the art at the time of the invention to use the hygromycin resistance gene in the methods of Pavlakis et al as an indicator to identify instability sequences (INS) of a gene encoding a transcript known or suspected to possess such INS sequences because Pavlakis et al teach it is within the skill of the art to use a gene encoding an mRNA which is expressed at relatively high levels (defined as being stable enough or highly expressed enough such that any decrease in the level of the mRNA or expressed protein can be detected by standard methods) as a reporter gene to identify such instability sequences and because Gritz et al teach that the gene encoding hygromycin B phosphotransferase is sufficiently well expressed that one can assay for its presence by direct genetic selection or by enzymatic assay in either prokaryotic or eukaryotic systems. One would have been motivated to do so in order to receive the expected benefit of being able to easily assay for protein encoded by the hybrid transcripts comprising the putative INS sequence, as taught by Pavlakis et al, by either enzymatic means or by direct genetic selection, as taught by Gritz et al. Absent any evidence to the contrary, there would have been a reasonable expectation of success in practicing the methods taught by Pavlakis et al with the hph gene, as taught by Gritz et al, to identify putative destabilizing INS regions in a desired gene transcript. In cases in which such a destabilizing element is identified using the hygromycin resistance gene as a reporter, one would necessarily have generated a short-lived transcript drug resistance gene.

The limitation of a cell transformed with the vector of claim 35 and selected with the corresponding drug does not necessarily confer any structural/functional distinction on the cell that would not be present in the cells taught by Schuler. Differences in gene expression for the

constructs taught in the specification appear to be due to the location of insertion of the targeting vector in the specification and not due to some structural aspect of the claimed vector itself.

Claims 34-38, 41-43 are rejected under 35 U.S.C. 103(a) as being unpatentable over Pavlakis et al (U.S. Patent No. 5,972,596; see the entire patent) in view of de la Luna et al (Gene 1988, Vol. 62, pages 121-126). **This rejection is maintained for reasons of record in Paper No. 14, mailed 2/12/03 and repeated below.**

The teachings of the Pavlakis et al patent ('596 patent) are described above and applied as before, except:

The Pavlakis et al patent does not explicitly teach an embodiment where the puromycin resistance gene is operatively linked to a coding sequence comprising an INS. The '596 patent doesn't explicitly teach the fusion of a coding sequence for puromycin resistance to any part of the c-fos gene.

The de la Luna et al reference teaches the construction and characterization of different vectors expressing the puromycin-resistance protein (puromycin-N-acetyl-transferase or PAC) for the efficient transformation of mammalian cells (e.g. Abstract). The de la Luna et al reference teaches that one can detect varying levels of PAC expressed from different constructs in COS-1 cells based upon an enzymatic assay (e.g. Table 1). The reference further teaches that one can detect different levels of PAC expression from the different expression constructs by genetic selection for stably transformed cells (e.g. Table II, expressed as the number of stable transformants per 10^6 recipient cells).

It would have been obvious to one of ordinary skill in the art at the time of the invention to use the puromycin resistance gene in the methods of Pavlakis et al as an indicator to identify instability sequences (INS) of a gene encoding a transcript known or suspected to possess such INS sequences because Pavlakis et al teach it is within the skill of the art to use a gene encoding an mRNA which is expressed at relatively high levels (defined as being stable enough or highly expressed enough such that any decrease in the level of the mRNA or expressed protein can be detected by standard methods) as a reporter gene to identify such instability sequences and because de la Luna et al teach that the gene encoding puromycin resistance is sufficiently well expressed that one can assay for its presence by genetic selection or by enzymatic assay. One would have been motivated to do so in order to receive the expected benefit of being able to easily assay for protein encoded by the hybrid transcripts comprising the putative INS sequence, as taught by Pavlakis et al, by either enzymatic means or by genetic selection, as taught by de la Luna et al. Absent any evidence to the contrary, there would have been a reasonable expectation of success in practicing the methods taught by Pavlakis et al with the puromycin resistance gene, as taught by de la Luna et al, to identify putative destabilizing INS regions in a desired gene transcript. In cases in which such a destabilizing element is identified using the puromycin resistance gene as a reporter, one would necessarily have generated a short-lived transcript drug resistance gene.

The limitation of a cell transformed with the vector of claim 35 and selected with the corresponding drug does not necessarily confer any structural/functional distinction on the cell that would not be present in the cells taught by Schuler. Differences in gene expression for the

constructs taught in the specification appear to be due to the location of insertion of the targeting vector in the specification and not due to some structural aspect of the claimed vector itself.

Response to Arguments

Applicant's arguments filed in Paper No. 17 have been fully considered but they are not persuasive. The response essentially argues: 1) the mere fact that two references can be combined does not make them obvious unless there is some desirability of that combination, 2), Pavlakis et al do not actually suggest the use of neomycin resistance gene, they only list it as a possible reporter gene, 3) Pavlakis et al do not actually reduce the neomycin construct to practice, 4) DePonti-Zilli do not teach an embodiment where their expression vector has an mRNA destabilizing sequence, 5) DePonti-Zilli et al were unsuccessful in showing that an mRNA destabilizing sequence could be characterized using such a vector, 6) de la Luna and Gritz et al are not analogous art.

Arguments that there is no motivation to combine the references used in the obviousness rejections are not persuasive because the primary reference explicity suggests the use of any reporter known in the art which can be easily assayed and specifically recites two of the embodiments that are claimed by applicants. The argument that there is no particular reason to choose the ones listed by Pavlakis et al as being embraced by their invention (e.g. neomycin) is not persuasive because Pavlakis et al do explicitly cite those embodiments as falling within the scope of their invention. With regard to the argument that Pavlakis et al never reduce the embodiments recited herein to practice in the form of an expression vector comprising a short-lived mRNA drug resistance gene, this argument is not persuasive in that Pavlakis et al merely have to be enabling at the time of filing of the instant application, in view of the art available at

the time of filing, for the 103 rejection made herein to be valid. If Pavlakis et al actually exemplified the claimed invention the rejection would necessarily have been made under 35 USC 102 instead.

Arguments that DePonti-Zilli et al were unsuccessful in characterizing an mRNA-destabilizing sequence are technically accurate, but misleading. DePonti-Zilli et al were successful in characterizing a sequence that had been proposed as possibly being a destabilizing sequence. By using an expression construct that is consistent with those taught by Pavlakis et al, the authors were able to demonstrate that the test sequence was not a destabilizing sequence. This is precisely the type of experiment taught by Pavlakis et al and the teachings of DePonti-Zilli et al conclusively demonstrate that one can use methods as taught by Pavlakis et al and including a neomycin resistance gene to characterize RNA sequences for their ability to stabilize/destabilize a particular sequence. Arguments that de la Luna et al and Gritz et al are non-analogous art, this assertion is inaccurate. Pavlakis et al teach their methods can be performed with an reporter for which assays are known, and suggest selectable markers (e.g. neomycin resistance gene) as one type of reporter. Gritz et al and de la Luna each teach assays for selectable markers and are, thus, analogous art.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 34-38, 41-43 are rejected under 35 U.S.C. 102(b) as being anticipated by Schuler et al (Cell, December 1988, Vol. 55, pages 1115-1122). **This is a new rejection.**

Schuler teach an experiment wherein coding sequences for neomycin resistance (neo) were operatively linked to untranslated regions obtained from genes encoding GM-CSF, c-fos, and c-myc. Shuler et al teach that the transcripts from these fusions had a shorter half-life following expression than the neomycin resistance transcript alone (e.g. Figure 4). The vectors taught by Schuler et al for expression of these genes are necessarily "expression" vectors. One of skill in the art would expect that the fusion transcripts described by Schuler et al would provide at least some degree of drug resistance as they encode the entire neomycin gene. The skilled artisan would also expect that one would be able to select cells in which expression is increase due to the presence of the destabilization sequences in the fusion transcripts because the fusion transcripts of Schuler et al do not appear to be structurally any different from those taught in the instant application.

The limitation of a cell transformed with the vector of claim 35 and selected with the corresponding drug does not necessarily confer any structural/functional distinction on the cell that would not be present in the cells taught by Schuler. Differences in gene expression for the constructs taught in the specification appear to be due to the location of insertion of the targeting vector in the specification and not due to some structural aspect of the claimed vector itself.

Because the Office does not have the facilities for examining and comparing the applicant's product with the products of the prior art, the burden is on the applicant to show a novel or unobvious difference between the claimed products and the products of the prior art (e.g. that the products of the prior art do not possess the same material structural and functional

characteristics of the claimed product). See in re Best, 562 F.2d 1252, 195 USPQ 430 (CCPA 1977).

Conclusion

No claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Gerald G Leffers Jr., PhD whose telephone number is (703) 308-6232. The examiner can normally be reached on 9:30am-6:00pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Remy Yucel can be reached on (703) 305-1998. The fax phone numbers for the organization where this application or proceeding is assigned are (703) 872-9306 for regular communications and (703) 872-9307 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is (703) 308-0196.

Gerald G Leffers Jr., PhD
Examiner
Art Unit 1636

Ggl
August 8, 2003